

spontaneous recurrent seizures (10–18 days), GAT-1 and GABA immunoreactivities were increased in the inner molecular layer by 6–9 days (the latent period) and persisted until periods of recurrent spontaneous seizures. Likewise, GAD immunoreactivity was increased in the inner molecular layer and, to a lesser degree, in the outer molecular layer at times ≥ 2 months following initial pilocarpine-induced status epilepticus. Studies performed at longer single time points following the initial insult also suggest axonal sprouting of GABAergic interneurons into granule cell dendritic regions. Increased GAD or GAT-1 immunoreactivity has been documented in the inner molecular layer in the kainic acid, electrical stimulation-induced status epilepticus, and pilocarpine models and in the outer molecular layer in the pilocarpine and electrical stimulation-induced status epilepticus models. Consistent with data obtained in animal models, increases in GAD immunoreactivity in the inner molecular layer and and GAT-1 immunoreactivity throughout the molecular layer have been documented in human epileptic hippocampus. Taken together, these data suggest an early loss of innervation followed by sprouting in GABAergic circuitry in the dendritic regions of dentate granule cells. The data further suggest that sprouting in GABAergic circuitry is an early event that precedes the appearance of spontaneous recurrent seizures.

Epilepsy treatment

- Block destabilising currents
- Increase stabilising currents
- Reduce synaptic excitation
 - Block glutamate release
 - Block glutamate receptors
- Increase synaptic inhibition
 - Increase GABA release
 - Potentiate GABA receptors

No anti-epileptic drugs can repair the network but they will prevent the occurrence of another seizure. There is no cure for epilepsy.

Blocking voltage gated Na-channels blocks depolarisations and drugs that do this include: phenytoin, carbamazepine, lamotrigine and sodium and valproate.

Blocking voltage gated Ca-channels blocks the release of neurotransmitters such as glutamate, drugs that do this includes: ethosuximide, gabapentin and phenytoin. Drugs that block glutamate block Na channels and Ca channels.